

## Mental Health: A Report of the Surgeon General

among others. Underlying this heterogeneous group of disorders is a state of heightened arousal or fear in relation to stressful events or feelings. The biological manifestations of anxiety, which are grounded in the "fight-or-flight" response, are unmistakable: they include surge in heart rate, sweating, and tensing of muscles. But this is certainly not the whole picture. Although the full array of biological causes and correlates of anxiety are not yet in our grasp, numerous effective treatments for anxiety disorders exist now. Treatment draws on an assortment of psychosocial and pharmacological approaches, administered alone or in combination.

Mood disorders take a monumental toll in human suffering, lost productivity, and suicide. Moreover, when unrecognized, they can result in unnecessary health care use. Mood disorders rank among the top 10 causes of *worldwide* disability (Murray & Lopez, 1996). Major depression and bipolar disorder are the most familiar mood disorders, but there are others including cyclothymia (alternating manic and depressive states that, while protracted, do not meet criteria for bipolar disorder) and dysthymia (a chronic, albeit symptomatically milder form of depression). The causes of mood disorders are not fully known. They may be triggered by stressful life events and enduring stressful social conditions (e.g., poverty and discrimination). With the exception of bipolar disorder, they too, like the anxiety disorders, are twice as common in women as men. One subtype of mood disorder, seasonal affective disorder, in which episodes of depression tend to occur in the late fall and winter, is seven times more common in women than in men (Blumenthal, 1988). Many psychosocial and genetic factors interact to dictate the appearance and persistence of mood disorders, according to the biopsychosocial model presented in Chapter 2.

Mood disorders, like anxiety disorders, can be treated with a host of effective pharmacological and psychosocial treatments. Either type of treatment is effective for about 50 to 70 percent of patients in outpatient settings (Depression Guideline Panel, 1993). Severe depression seems to resolve more quickly with pharmacotherapy (Depression Guideline Panel, 1993)

and may be helped further by multimodal therapy (the combination of pharmacotherapy and psychotherapy) (Thase et al., 1997b). Despite the efficacy of treatment, a surprising fraction of those with mood disorders go untreated (Katon et al., 1992; Narrow et al., 1993; Wells et al., 1994; Thase, 1996). The foremost barriers to treatment include cost, stigma, and problems in the organization of service systems that contribute to the underrecognition of these disorders.

Schizophrenia affects about 1 percent of the population, yet its severity and persistence reverberate throughout the mental health service system. Schizophrenia is marked by profound alterations in cognition and emotion. Symptoms frequently include hearing internal voices or experiencing other sensations not connected to an obvious source (hallucinations) and assigning unusual significance or meaning to normal events or holding false personal beliefs (delusions). The course of illness in schizophrenia is quite variable, with most people having periods of exacerbation and remission. Schizophrenia had once been thought to have a uniformly downhill course, but recent research dispels this view. Long-term followup studies show that many individuals with schizophrenia significantly improve and some recover (Ciompi, 1980; Harding et al., 1992). Although the causes of schizophrenia are not fully known, research points to the prominent role of genetic factors and to the impact of adverse environmental influences during early brain development (Tsuang et al., 1991; Weinberger & Lipska, 1995; Andreasen, 1997b). New pharmacological treatments are at least as effective as past pharmacological treatments with fewer troubling side effects.

Effective treatment of schizophrenia extends well beyond pharmacological therapy: it also includes psychosocial interventions, family interventions, and vocational and psychosocial rehabilitation. For those patients who are high service users, treatment should be coordinated by an interdisciplinary team that provides high-intensity, community-based services (Lehman & Steinwachs, 1998a). The prototype for this intensive case-management approach, which is useful for persons with other severe and persistent mental disorders as

well, is assertive community treatment, described more thoroughly later in this chapter. Among the services included in this approach is substance abuse treatment. Its inclusion stems from findings that about *half* of patients with serious mental disorders (including schizophrenia) develop alcohol or other drug abuse problems (Drake & Osher, 1997). Even though research generated a range of recommendations for effective treatment of schizophrenia, it is alarming that less than 50 percent of patients actually receive many of the recommended treatments and that the gap was more pronounced in African Americans (Lehman & Steinwachs, 1998b).

The social consequences of serious mental disorders—family disruption, loss of employment and housing—can be calamitous. Comprehensive treatment, which includes services that exist outside the formal treatment system, is crucial to ameliorate symptoms, assist recovery, and, to the extent that these efforts are successful, redress stigma. Consumer self-help programs, family self-help, advocacy, and services for housing and vocational assistance complement and supplement the formal treatment system. Many of these services are operated by consumers, that is, people who use mental health services themselves. The logic behind their leadership in delivery of these services is that consumers are thought to be capable of engaging others with mental disorders, serving as role models, and increasing the sensitivity of service systems to the needs of people with mental disorders (Mowbray et al., 1996).

### Mental Health in Adulthood

What constitutes mental health during the adult years? A widely used standard of mental health is the *absence* of a defined mental disorder. This standard has its limitations (discussed later), yet remains useful for epidemiological purposes. Epidemiology studies investigate the prevalence of mental disorders within several time frames: current, the past 12 months, and across a lifetime. Two well-designed national epidemiologic surveys estimate that about 80 percent of the adult population of the United States do not have a mental disorder during a year and hence may be

considered “mentally healthy” (i.e., absence of a mental disorder) during any given year (Regier et al., 1993; Kessler et al., 1994). Thus, the popular notion that *everyone* is “dysfunctional” is far from the truth (Table 4-1). Yet, from time to time, many adults experience mental health problems.

Defining mental health by the absence of mental disorder does not convey the full picture of mental health. Among its limitations, this definition excludes adults with mental disorders who function well between episodes of illness. These people often are considered by themselves, and by coworkers, friends, and families, to be “mentally healthy” in spite of a history of mental illness and the risk of recurrence.

In addition to the mental health criteria cited earlier—that is, the successful performance of mental function, enabling individuals to cope with adversity and to flourish in their education, vocation, and personal relationships—a complementary approach defines the positive features of mental health in terms of attaining developmental milestones of adulthood, or in terms of displaying selected personality characteristics, traits, or attributes. Developmental theorist Erik Erikson viewed mental health in adulthood as achieving developmental tasks or milestones. According to Erikson’s formulation and his subsequent empirical research on adult men, adulthood was the time for overcoming what he termed “psychosocial crises,” the resolution of which led to satisfactory interpersonal and sexual relationships and to the pursuit of broader concerns for society and future generations (Erikson, 1963; Vaillant, 1977). However, these milestones, and the developmental theories that underpin them, have been criticized as reflecting the norms of European males rather than of women and other cultures.

### Personality Traits

Mental health and mental illness can be seen as the product of various personality traits, behavior patterns, and other characteristics which have roots in the individual’s prior life experiences or biology.

## Mental Health: A Report of the Surgeon General

**Table 4-1. Best estimate 1-year prevalence based on ECA and NCS, ages 18–54**

	ECA Prevalence (%)	NCS Prevalence (%)	Best Estimate ** (%)
Any Anxiety Disorder	13.1	18.7	16.4
Simple Phobia	8.3	8.6	8.3
Social Phobia	2.0	7.4	2.0
Agoraphobia	4.9	3.7	4.9
GAD	(1.5)*	3.4	3.4
Panic Disorder	1.6	2.2	1.6
OCD	2.4	(0.9)*	2.4
PTSD	(1.9)*	3.6	3.6
Any Mood Disorder	7.1	11.1	7.1
MD Episode	6.5	10.1	6.5
Unipolar MD	5.3	8.9	5.3
Dysthymia	1.6	2.5	1.6
Biopolar I	1.1	1.3	1.1
Biopolar II	0.6	0.2	0.6
Schizophrenia	1.3	—	1.3
Nonaffective Psychosis	—	0.2	0.2
Somatization	0.2	—	0.2
ASP	2.1	—	2.1
Anorexia Nervosa	0.1	—	0.1
Severe Cognitive Impairment	1.2	—	1.2
Any Disorder	19.5	23.4	21.0

\*Numbers in parentheses indicate the prevalence of the disorder without any comorbidity. These rates were calculated using the NCS data for GAD and PTSD, and the ECA data for OCD. The rates were not used in calculating the any anxiety disorder and any disorder totals for the ECA and NCS columns. The unduplicated GAD and PTSD rates were added to the best estimate total for any anxiety disorder (3.3%) and any disorder (1.5%).

\*\*In developing best-estimate 1-year prevalence rates from the two studies, a conservative procedure was followed that had previously been used in an independent scientific analysis comparing these two data sets (Andrews, 1995). For any mood disorder and any anxiety disorder, the lower estimate of the two surveys was selected, which for these data was the ECA. The best estimate rates for the individual mood and anxiety disorders were then chosen from the ECA only, in order to maintain the relationships between the individual disorders. For other disorders that were not covered in both surveys, the available estimate was used.

Key to abbreviations: ECA, Epidemiologic Catchment Area; NCS, National Comorbidity Study; GAD, generalized anxiety disorder; OCD, obsessive-compulsive disorder; PTSD, post-traumatic stress disorder; MD, major depression; ASP, antisocial personality disorder.

Source: D. Regier, W. Narrow, & D. Rae, personal communication, 1999

Personality traits are thought to confer either beneficial or detrimental effects on mental health during adulthood. Here too, however, there may be insufficient attention to gender and culture. The culture-bound nature of much of behavior has limited widespread predictive validity of personality research (Mischel & Shoda, 1968). With this caveat in mind, a brief summary of healthy and maladaptive characteristics follows.

### *Self-Esteem*

Self-esteem refers to an abiding set of beliefs about one's own worth, competence, and abilities to relate to others (Vaughan & Oldham, 1997). Self-esteem also has been conceptualized as buffering the individual from adverse life events. Emotional well-being is often associated with a slightly positive, yet realistic, outlook (Alloy & Abramson, 1988). The opposite outlook is

characterized by pessimism, demoralization, or minor symptoms of anxiety and depression. One seminal aspect of self-esteem has garnered much research attention: self-efficacy (Bandura, 1977). Self-efficacy is defined as confidence in one's own abilities to cope with adversity, either independently or by obtaining appropriate assistance from others. Self-efficacy is a major component of the construct known as resilience (i.e., the ability to withstand and overcome adversity). Other components of resilience include intelligence and problem solving, although resilience is also facilitated by having adequate social support (Beardslee & Vaillant, 1997).

### *Neuroticism*

Neuroticism is a construct that refers to a broad pattern of psychological, emotional, and psychophysiologic reactivity (Eysenck & Eysenck, 1975). The opposite of neuroticism is stability or equanimity, which are major components of mental health. A high level of neuroticism is associated with a predisposition toward recognizing the dangerous, harmful, or defeating aspects of a situation and the tendency to respond with worry, anticipatory anxiety, emotionality, pessimism, and dissatisfaction. Neuroticism is associated with a greater risk of early-onset depressive and anxiety disorders (Clark et al., 1994). Neuroticism also may be linked to a particular cognitive attributional style in which life events are perceived to be large in impact and more difficult to change (Alloy et al., 1984). For example, this attributional style is embodied by pessimists who see every setback or failure as lasting forever, undermining everything, and being their fault (Seligman, 1991). Neuroticism also is associated with more rigid or distorted attitudes and beliefs about one's competence (Beck, 1976).

### *Avoidance*

Avoidance describes an exaggerated predisposition to withdraw from novel situations and to avoid personal challenges as threats. This is the behavioral state that often accompanies the distress of someone who has a high level of neuroticism and low self-efficacy (Vaughan & Oldham, 1997). Closely related to the

characteristics of behavioral inhibition or introversion, the trait of avoidance appears to be partly inherited and is associated with shyness, anxiety, and depressive disorders in both childhood and adult life, as well as the subsequent development of substance abuse disorders (Vaughan & Oldham, 1997; Kagan et al., 1988). The people with low levels of harm avoidance are described as "healthy extroverts" and are characterized by confident, carefree, or outgoing behaviors.

### *Impulsivity*

Impulsivity is a trait that is associated with poor modulation of emotions, especially anger, difficulty delaying gratification, and novelty seeking. There is some developmental continuity between high levels of impulsivity in childhood and several adult mental disorders, including attention deficit hyperactivity disorder, bipolar disorder, and substance abuse disorders (Svrakic et al., 1993; Rothbart & Ahadi, 1994). Impulsivity also is associated with physical abuse (both as victim and, subsequently, as perpetrator) and antisocial personality traits (Vaughan & Oldham, 1997).

### *Sociopathy*

This set of traits and behaviors refers to the predisposition to engage in dishonest, hurtful, unfaithful, and at times dangerous conduct to benefit one's own ends. The opposite of sociopathy may be referred to as character or scrupulosity. In its full form, sociopathy is referred to as antisocial personality disorder (DSM-IV). Sociopathy is characterized by a tendency and ability to disregard laws and rules, difficulties reciprocating within empathic and intimate relationships, less internalization of moral standards (i.e., a weaker conscience or superego), and an insensitivity to the needs and rights of others. People scoring high in sociopathy often have problems with aggressivity and are overrepresented among criminal populations. Although not invariably associated with criminality, sociopathy is associated with problematic, unethical, and morally questionable conduct in the workplace and within social systems. Marked sociopathy is much more common among men than

## Mental Health: A Report of the Surgeon General

women, although several other disorders (borderline and histrionic personality disorders and somatization disorder) are overrepresented among women within the same families (Widiger & Costa, 1994).

In summary, the various traits and behavioral patterns that epitomize strong mental health do not, of course, exist in a vacuum: they develop in a social context, and they underpin people's ability to handle psychological and social adversity and the exposure to stressful life events. Furthermore, as reviewed in Chapter 3, severe or repeated trauma during youth may have enduring effects on both neurobiological and psychological development, altering stress responsivity and adult behavior patterns. Perhaps the best documented evidence of such enduring effects has been shown in young adults who experienced severe sexual or physical abuse in childhood. These individuals experience a greatly increased risk of mood, anxiety, and personality disorders throughout adult life.

### Stressful Life Events

The most common psychological and social stressors in adult life include the breakup of intimate romantic relationships, death of a family member or friend, economic hardships, racism and discrimination, poor physical health, and accidental and intentional assaults on physical safety (Holmes & Rahe, 1967; Lazarus & Folkman, 1984; Kreiger et al., 1993). Although some stressors are so powerful that they would evoke significant emotional distress in most otherwise mentally healthy people, the majority of stressful life events do not invariably trigger mental disorders. Rather, they are more likely to spawn mental disorders in people who are vulnerable biologically, socially, and/or psychologically (Lazarus & Folkman, 1984; Brown & Harris, 1989; Kendler et al., 1995). Understanding variability among individuals to a stressful life event is a major challenge to research. Groups at greater statistical risk include women, young and unmarried people, African Americans, and individuals with lower socioeconomic status (Ulbrich et al., 1989; McLeod & Kessler, 1990; Turner et al., 1995; Miranda & Green, 1999).

Divorce is a common example. Approximately one-half of all marriages now end in divorce, and about 30 to 40 percent of those undergoing divorce report a significant increase in symptoms of depression and anxiety (Brown & Harris, 1989). Vulnerability to depression and anxiety is greater among those with a personal history of mental disorders earlier in life and is lessened by strong social support. For many, divorce conveys additional economic adversities and the stress of single parenting. Single mothers face twice the risk of depression as do married mothers (Brown & Moran, 1997).

The death of a child or spouse during early or midadult life is much less common than divorce but generally is of greater potency in provoking emotional distress (Kim & Jacobs, 1995). Rates of diagnosable mental disorders during periods of grief are attenuated by the convention not to diagnose depression during the first 2 months of bereavement (Clayton & Darvish, 1979). In fact, people are generally unlikely to seek professional treatment during bereavement unless the severity of the emotional and behavioral disturbance is incapacitating.

A majority of Americans never will confront the stress of surviving a severe, life-threatening accident or physical assault (e.g., mugging, robbery, rape); however, some segments of the population, particularly urban youths and young adults, have exposure rates as high as 25 to 30 percent (Helzer et al., 1987; Breslau et al., 1991). Life-threatening trauma frequently provokes emotional and behavioral reactions that jeopardize mental health. In the most fully developed form, this syndrome is called post-traumatic stress disorder (DSM-IV), which is described later in this chapter. Women are twice as likely as men to develop post-traumatic stress disorder following exposure to life-threatening trauma (Breslau et al., 1998.)

More familiar to many Americans is the chronic strain that poor physical health and relationship problems place on day-to-day well-being. Relationship problems include unsatisfactory intimate relationships; conflicted relationships with parents, siblings, and children; and "falling-out" with coworkers, friends, and

neighbors. In mid-adult life, the stress of caretaking for elderly parents also becomes more common.

Relationship problems at least double the risk of developing a mental disorder, although they are less immediately threatening or potentially cataclysmic than divorce or the death of a spouse or child (Brown & Harris, 1989). Finally, cumulative adversity appears to be more potent than stressful events in isolation as a predictor of psychological distress and mental disorders (Turner & Lloyd, 1995).

### ***Past Trauma and Child Sexual Abuse***

Severe trauma in childhood may have enduring effects into adulthood (Browne & Finkelhor, 1986). Past trauma includes sexual and physical abuse, and parental death, divorce, psychopathology, and substance abuse (reviewed in Turner & Lloyd, 1995).

Child sexual abuse is one of the most common stressors, with effects that persist into adulthood. It disproportionately affects females. Although definitions are still evolving, child sexual abuse is often defined as forcible touching of breasts or genitals or forcible intercourse (including anal, oral, or vaginal sex) before the age of 16 or 18 (Goodman et al., 1997). Epidemiology studies of adults in varying segments of the community have found that 15 to 33 percent of females and 13 to 16 percent of males were sexually abused in childhood (Polusny & Follette, 1995). A recent, large epidemiological study of adults in the general community found a lower prevalence (12.8 percent for females and 4.3 percent for males); however, the definition of sexual abuse was more restricted than in past studies (MacMillan et al., 1997). Sexual abuse in childhood has a mean age of onset estimated at 7 to 9 years of age (Polusny & Follette, 1995). In over 25 percent of cases of child sexual abuse, the offense was committed by a parent or parent substitute (Sedlak & Broadhurst, 1996).

The long-term consequences of past childhood sexual abuse are profound, yet vary in expression. They range from depression and anxiety to problems with social functioning and adult interpersonal relationships (Polusny & Follette, 1995). Post-traumatic stress disorder is a common sequela, found in 33 to 86

percent of adult survivors of child sexual abuse (Polusny & Follette, 1995). In a recent review, Weiss et al. (1999) found that sexual abuse was a specific risk factor for adult-onset depression and twice as many women as men reported a history of abuse. Other long-term effects include self-destructive behavior, social isolation, poor sexual adjustment, substance abuse, and increased risk of revictimization (Browne & Finkelhor, 1986; Briere, 1992).

Very few treatments specifically for adult survivors of childhood abuse have been studied in randomized controlled trials (IOM, 1998). Group therapy and Interpersonal Transaction group therapy were found to be more effective for female survivors than an experimental control condition that offered a less appropriate intervention (Alexander et al., 1989, 1991). In the practice setting, most psychosocial and pharmacological treatments are tailored to the primary diagnosis, which, as noted above, varies widely and may not attend to the special needs of those also reporting abuse history.

### ***Domestic Violence***

Domestic violence is a serious and startlingly common public health problem with mental health consequences for victims, who are overwhelmingly female, and for children who witness the violence. Domestic violence (also known as intimate partner violence) features a pattern of physical and sexual abuse, psychological abuse with verbal intimidation, and/or social isolation or deprivation. Estimates are that 8 to 17 percent of women are victimized annually in the United States (Wilt & Olsen, 1996). Pinpointing the prevalence is hindered by variations in the way domestic violence is defined and by problems in detection and underreporting. Women are often fearful that their reporting of domestic violence will precipitate retaliation by the batterer, a fear that is not unwarranted (Sisley et al., 1999).

Victims of domestic violence are at increased risk for mental health problems and disorders as well as physical injury and death. Domestic violence is considered one of the foremost causes of serious injury to women ages 15 to 44, accounting for about 30

## Mental Health: A Report of the Surgeon General

percent of all acute injuries to women seen in emergency departments (Wilt & Olsen, 1996). According to the U.S. Department of Justice, females were victims in about 75 percent of the almost 2,000 homicides between intimates in 1996 (cited in Sisley et al., 1999). The mental health consequences of domestic violence include depression, anxiety disorders (e.g., post-traumatic stress disorder), suicide, eating disorders, and substance abuse (IOM, 1998; Eisenstat & Bancroft, 1999). Children who witness domestic violence may suffer acute and long-term emotional disturbances, including nightmares, depression, learning difficulties, and aggressive behavior. Children also become at risk for subsequent use of violence against their dating partners and wives (el-Bayoumi et al., 1998; NRC, 1998; Sisley et al., 1999).

Mental health interventions for victims, children, and batterers are highly important. Individual counseling and peer support groups are the interventions most frequently used by battered women. However, there is a lack of carefully controlled, methodologically robust studies of interventions and their outcomes, according to a report by the Institute of Medicine and National Research Council (IOM, 1998). A research agenda for violence against women was developed (IOM, 1996) and has served as an impetus for an ongoing research program sponsored by the U.S. Departments of Justice and Health and Human Services. Clearly, there is an urgent need for development and rigorous evaluation of prevention programs to safeguard against intimate partner violence and its impact on children.

### *Interventions for Stressful Life Events*

Stressful life events, even for those at the peak of mental health, erode quality of life and place people at risk for symptoms and signs of mental disorders. There is an ever-expanding list of formal and informal interventions to aid individuals coping with adversity. Sources of informal interventions include family and friends, education, community services, self-help groups, social support networks, religious and spiritual endeavors, complementary healers, and physical activities. As valuable as these activities may be for

promoting mental health, they have received less research attention than have interventions for mental disorders. Nevertheless, there are selected interventions to help people cope with stressors, such as bereavement programs and programs for caregivers (see Chapter 5) as well as couples therapy and physical activity.

Couples therapy is the umbrella term applied to interventions that aid couples in distress. The best studied interventions are behavioral couples therapy, cognitive-behavioral couples therapy, and emotion-focused couples therapy. A recent review article evaluated the body of evidence on the effectiveness of couples therapy and programs to prevent marital discord (Christensen & Heavey, 1999). The review found that about 65 percent of couples in therapy did improve, whereas 35 percent of control couples also improved. Couples therapy ameliorates relationship distress and appears to alleviate depression. The gains from couples therapy generally last through 6 months, but there are few long-term assessments (Christensen & Heavey, 1999). Similarly, interventions to prevent marital discord yield short-term improvements in marital adjustment and stability, but there is insufficient study of long-term outcomes. The prevention programs receiving the most study are the Couple Communication Program, Relationship Enhancement, and the Prevention and Relationship Enhancement Program (Christensen & Heavey, 1999). Greater research is needed to overcome gaps in knowledge and to extend findings to a broader array of programs, to diverse populations of couples, and to a wider set of outcomes, including effects on children.

Physical activities are a means to enhance somatic health as well as to deal with stress. A recent Surgeon General's Report on Physical Activity and Health evaluated the evidence for physical activities serving to enhance mental health (U.S. Department of Health and Human Services [DHHS], 1996). Aerobic physical activities, such as brisk walking and running, were found to improve mental health for people who report *symptoms* of anxiety and depression and for those who are diagnosed with some forms of depression. The mental health benefits of physical activity for individuals in relatively good physical and mental

health were not as evident, but the studies did not have sufficient rigor from which to draw unequivocal conclusions (DHHS, 1996).

### **Prevention of Mental Disorders**

A promising development in prevention of a specific mental disorder in adults occurred with the publication of results from the San Francisco Depression Research Project (Munoz et al., 1995). This study investigated 150 primary care patients who did not meet diagnostic criteria for depression and who were being seen in a public clinic for other problems. They were randomized to either psychoeducation—an 8-week cognitive behavioral course to help them control and manage moods—or to a control condition. One year later, those who received psychoeducation were found to have developed significantly fewer depression symptoms than members of the control group. This trial is noteworthy in two major respects: it was a randomized controlled trial and its participants were low-income individuals, with high representation of all major minority groups. Low-income individuals are considered a high-risk population because of studies documenting their higher prevalence of mental disorders. This study demonstrated in a methodologically rigorous fashion that depression may be preventable in some cases. It serves as a model for extending the concept of prevention to many mental disorders. Prevention research is vitally important and needs to be enhanced.

### **Anxiety Disorders**

The anxiety disorders are the most common, or frequently occurring, mental disorders. They encompass a group of conditions that share extreme or pathological anxiety as the principal disturbance of mood or emotional tone. Anxiety, which may be understood as the pathological counterpart of normal fear, is manifest by disturbances of mood, as well as of thinking, behavior, and physiological activity.

#### **Types of Anxiety Disorders**

The anxiety disorders include panic disorder (with and without a history of agoraphobia), agoraphobia (with

and without a history of panic disorder), generalized anxiety disorder, specific phobia, social phobia, obsessive-compulsive disorder, acute stress disorder, and post-traumatic stress disorder (DSM-IV). In addition, there are adjustment disorders with anxious features, anxiety disorders due to general medical conditions, substance-induced anxiety disorders, and the residual category of anxiety disorder not otherwise specified (DSM-IV).

Anxiety disorders not only are common in the United States, but they are ubiquitous across human cultures (Regier et al., 1993; Kessler et al., 1994; Weissman et al., 1997). In the United States, 1-year prevalence for all anxiety disorders among adults ages 18 to 54 exceeds 16 percent (Table 4-1), and there is significant overlap or comorbidity with mood and substance abuse disorders (Regier et al., 1990; Goldberg & Lecrubier, 1995; Magee et al., 1996). The longitudinal course of these disorders is characterized by relatively early ages of onset, chronicity, relapsing or recurrent episodes of illness, and periods of disability (Keller & Hanks, 1994; Gorman & Coplan, 1996; Liebowitz, 1997; Marcus et al., 1997). Although few psychological autopsy studies of adult suicides have included a focus on comorbid conditions (Conwell & Brent, 1995), it is likely that the rate of comorbid anxiety in suicide is underestimated. Panic disorder and agoraphobia, particularly, are associated with increased risks of attempted suicide (Hornig & McNally, 1995; American Psychiatric Association, 1998).

#### ***Panic Attacks and Panic Disorder***

A panic attack is a discrete period of intense fear or discomfort that is associated with numerous somatic and cognitive symptoms (DSM-IV). These symptoms include palpitations, sweating, trembling, shortness of breath, sensations of choking or smothering, chest pain, nausea or gastrointestinal distress, dizziness or lightheadedness, tingling sensations, and chills or blushing and “hot flashes.” The attack typically has an abrupt onset, building to maximum intensity within 10 to 15 minutes. Most people report a fear of dying, “going crazy,” or losing control of emotions or behavior. The experiences generally provoke a strong



## Mental Health: A Report of the Surgeon General

urge to escape or flee the place where the attack begins and, when associated with chest pain or shortness of breath, frequently results in seeking aid from a hospital emergency room or other type of urgent assistance. Yet an attack rarely lasts longer than 30 minutes. Current diagnostic practice specifies that a panic attack must be characterized by at least four of the associated somatic and cognitive symptoms described above. The panic attack is distinguished from other forms of anxiety by its intensity and its sudden, episodic nature. Panic attacks may be further characterized by the relationship between the onset of the attack and the presence or absence of situational factors. For example, a panic attack may be described as *unexpected*, *situationally bound*, or *situationally predisposed* (usually, but not invariably occurring in a particular situation). There are also attenuated or "limited symptom" forms of panic attacks.

Panic attacks are not always indicative of a mental disorder, and up to 10 percent of otherwise healthy people experience an isolated panic attack per year (Barlow, 1988; Klerman et al., 1991). Panic attacks also are not limited to panic disorder. They commonly occur in the course of social phobia, generalized anxiety disorder, and major depressive disorder (DSM-IV).

Panic disorder is diagnosed when a person has experienced at least two unexpected panic attacks *and* develops persistent concern or worry about having further attacks or changes his or her behavior to avoid or minimize such attacks. Whereas the number and severity of the attacks varies widely, the concern and avoidance behavior are essential features. The diagnosis is inapplicable when the attacks are presumed to be caused by a drug or medication or a general medical disorder, such as hyperthyroidism.

Lifetime rates of panic disorder of 2 to 4 percent and 1-year rates of about 2 percent are documented consistently in epidemiological studies (Kessler et al., 1994; Weissman et al., 1997) (Table 4-1). Panic disorder is frequently complicated by major depressive disorder (50 to 65 percent lifetime comorbidity rates) and alcoholism and substance abuse disorders (20 to 30 percent comorbidity) (Keller & Hanks, 1994; Magee et

al., 1996; Liebowitz, 1997). Panic disorder is also concomitantly diagnosed, or co-occurs, with other specific anxiety disorders, including social phobia (up to 30 percent), generalized anxiety disorder (up to 25 percent), specific phobia (up to 20 percent), and obsessive-compulsive disorder (up to 10 percent) (DSM-IV). As discussed subsequently, approximately one-half of people with panic disorder at some point develop such severe avoidance as to warrant a separate description, panic disorder with agoraphobia.

Panic disorder is about twice as common among women as men (American Psychiatric Association, 1998). Age of onset is most common between late adolescence and midadult life, with onset relatively uncommon past age 50. There is developmental continuity between the anxiety syndromes of youth, such as separation anxiety disorder. Typically, an early age of onset of panic disorder carries greater risks of comorbidity, chronicity, and impairment. Panic disorder is a familial condition and can be distinguished from depressive disorders by family studies (Rush et al., 1998).

### Agoraphobia

The ancient term agoraphobia is translated from Greek as fear of an open marketplace. Agoraphobia today describes severe and pervasive anxiety about being in situations from which escape might be difficult or avoidance of situations such as being alone outside of the home, traveling in a car, bus, or airplane, or being in a crowded area (DSM-IV).

Most people who present to mental health specialists develop agoraphobia after the onset of panic disorder (American Psychiatric Association, 1998). Agoraphobia is best understood as an adverse behavioral outcome of repeated panic attacks and the subsequent worry, preoccupation, and avoidance (Barlow, 1988). Thus, the formal diagnosis of panic disorder with agoraphobia was established. However, for those people in communities or clinical settings who do not meet full criteria for panic disorder, the formal diagnosis of agoraphobia without history of panic disorder is used (DSM-IV).

The 1-year prevalence of agoraphobia is about 5 percent (Table 4-1). Agoraphobia occurs about two times more commonly among women than men (Magee et al., 1996). The gender difference may be attributable to social-cultural factors that encourage, or permit, the greater expression of avoidant coping strategies by women (DSM-IV), although other explanations are possible.

### ***Specific Phobias***

These common conditions are characterized by marked fear of specific objects or situations (DSM-IV). Exposure to the object of the phobia, either in real life or via imagination or video, invariably elicits intense anxiety, which may include a (situationally bound) panic attack. Adults generally recognize that this intense fear is irrational. Nevertheless, they typically avoid the phobic stimulus or endure exposure with great difficulty. The most common specific phobias include the following feared stimuli or situations: animals (especially snakes, rodents, birds, and dogs); insects (especially spiders and bees or hornets); heights; elevators; flying; automobile driving; water; storms; and blood or injections.

Approximately 8 percent of the adult population suffers from one or more specific phobias in 1 year (Table 4-1). Much higher rates would be recorded if less rigorous diagnostic requirements for avoidance or functional impairment were employed. Typically, the specific phobias begin in childhood, although there is a second "peak" of onset in the middle 20s of adulthood (DSM-IV). Most phobias persist for years or even decades, and relatively few remit spontaneously or without treatment.

The specific phobias generally do not result from exposure to a single traumatic event (i.e., being bitten by a dog or nearly drowning) (Marks, 1969). Rather, there is evidence of phobia in other family members and social or vicarious learning of phobias (Cook & Mineka, 1989). Spontaneous, unexpected panic attacks also appear to play a role in the development of specific phobia, although the particular pattern of avoidance is much more focal and circumscribed.

### ***Social Phobia***

Social phobia, also known as social anxiety disorder, describes people with marked and persistent anxiety in social situations, including performances and public speaking (Ballenger et al., 1998). The critical element of the fearfulness is the possibility of embarrassment or ridicule. Like specific phobias, the fear is recognized by adults as excessive or unreasonable, but the dreaded social situation is avoided or is tolerated with great discomfort. Many people with social phobia are preoccupied with concerns that others will see their anxiety symptoms (i.e., trembling, sweating, or blushing); or notice their halting or rapid speech; or judge them to be weak, stupid, or "crazy." Fears of fainting, losing control of bowel or bladder function, or having one's mind going blank are also not uncommon. Social phobias generally are associated with significant anticipatory anxiety for days or weeks before the dreaded event, which in turn may further handicap performance and heighten embarrassment.

The 1-year prevalence of social phobia ranges from 2 to 7 percent (Table 4-1), although the lower figure probably better captures the number of people who experience significant impairment and distress. Social phobia is more common in women (Wells et al., 1994). Social phobia typically begins in childhood or adolescence and, for many, it is associated with the traits of shyness and social inhibition (Kagan et al., 1988). A public humiliation, severe embarrassment, or other stressful experience may provoke an intensification of difficulties (Barlow, 1988). Once the disorder is established, complete remissions are uncommon without treatment. More commonly, the severity of symptoms and impairments tends to fluctuate in relation to vocational demands and the stability of social relationships. Preliminary data suggest social phobia to be familial (Rush et al., 1998).

### ***Generalized Anxiety Disorder***

Generalized anxiety disorder is defined by a protracted ( $\geq 6$  months' duration) period of anxiety and worry, accompanied by multiple associated symptoms (DSM-IV). These symptoms include muscle tension, easy fatiguability, poor concentration, insomnia, and

## Mental Health: A Report of the Surgeon General

irritability. In youth, the condition is known as overanxious disorder of childhood. In DSM-IV, an essential feature of generalized anxiety disorder is that the anxiety and worry cannot be attributable to the more focal distress of panic disorder, social phobia, obsessive-compulsive disorder, or other conditions. Rather, as implied by the name, the excessive worries often pertain to many areas, including work, relationships, finances, the well-being of one's family, potential misfortunes, and impending deadlines. Somatic anxiety symptoms are common, as are sporadic panic attacks.

Generalized anxiety disorder occurs more often in women, with a sex ratio of about 2 women to 1 man (Brawman-Mintzer & Lydiard, 1996). The 1-year population prevalence is about 3 percent (Table 4-1). Approximately 50 percent of cases begin in childhood or adolescence. The disorder typically runs a fluctuating course, with periods of increased symptoms usually associated with life stress or impending difficulties. There does not appear to be a specific familial association for general anxiety disorder. Rather, rates of other mood and anxiety disorders typically are greater among first-degree relatives of people with generalized anxiety disorder (Kendler et al., 1987).

### ***Obsessive-Compulsive Disorder***

Obsessions are recurrent, intrusive thoughts, impulses, or images that are perceived as inappropriate, grotesque, or forbidden (DSM-IV). The obsessions, which elicit anxiety and marked distress, are termed "ego-alien" or "ego-dystonic" because their content is quite unlike the thoughts that the person usually has. Obsessions are perceived as uncontrollable, and the sufferer often fears that he or she will lose control and act upon such thoughts or impulses. Common themes include contamination with germs or body fluids, doubts (i.e., the worry that something important has been overlooked or that the sufferer has unknowingly inflicted harm on someone), order or symmetry, or loss of control of violent or sexual impulses.

Compulsions are repetitive behaviors or mental acts that reduce the anxiety that accompanies an obsession or "prevent" some dreaded event from happening (DSM-IV). Compulsions include both overt behaviors, such as hand washing or checking, and mental acts including counting or praying. Not uncommonly, compulsive rituals take up long periods of time, even hours, to complete. For example, repeated hand washing, intended to remedy anxiety about contamination, is a common cause of contact dermatitis.

Although once thought to be rare, obsessive-compulsive disorder has now been documented to have a 1-year prevalence of 2.4 percent (Table 4-1). Obsessive-compulsive disorder is equally common among men and women.

Obsessive-compulsive disorder typically begins in adolescence to young adult life (males) or in young adult life (females) (Burke et al., 1990; DSM-IV). For most, the course is fluctuating and, like generalized anxiety disorder, symptom exacerbations are usually associated with life stress. Common comorbidities include major depressive disorder and other anxiety disorders. Approximately 20 to 30 percent of people in clinical samples with obsessive-compulsive disorder report a past history of tics, and about one-quarter of these people meet the full criteria for Tourette's disorder (DSM-IV). Conversely, up to 50 percent of people with Tourette's disorder develop obsessive-compulsive disorder (Pitman et al., 1987).

Obsessive-compulsive disorder has a clear familial pattern and somewhat greater familial specificity than most other anxiety disorders. Furthermore, there is an increased risk of obsessive-compulsive disorder among first-degree relatives with Tourette's disorder. Other mental disorders that may fall within the spectrum of obsessive-compulsive disorder include trichotillomania (compulsive hair pulling), compulsive shoplifting, gambling, and sexual behavior disorders (Hollander, 1996). The latter conditions are somewhat discrepant because the compulsive behaviors are less ritualistic and yield some outcomes that are pleasurable or

gratifying. Body dysmorphic disorder is a more circumscribed condition in which the compulsive and obsessive behavior centers around a preoccupation with one's appearance (i.e., the syndrome of imagined ugliness) (Phillips, 1991).

### **Acute and Post-Traumatic Stress Disorders**

Acute stress disorder refers to the anxiety and behavioral disturbances that develop within the first month after exposure to an extreme trauma. Generally, the symptoms of an acute stress disorder begin during or shortly following the trauma. Such extreme traumatic events include rape or other severe physical assault, near-death experiences in accidents, witnessing a murder, and combat. The symptom of dissociation, which reflects a perceived detachment of the mind from the emotional state or even the body, is a critical feature. Dissociation also is characterized by a sense of the world as a dreamlike or unreal place and may be accompanied by poor memory of the specific events, which in severe form is known as dissociative amnesia. Other features of an acute stress disorder include symptoms of generalized anxiety and hyperarousal, avoidance of situations or stimuli that elicit memories of the trauma, and persistent, intrusive recollections of the event via flashbacks, dreams, or recurrent thoughts or visual images.

If the symptoms and behavioral disturbances of the acute stress disorder persist for more than 1 month, and if these features are associated with functional impairment or significant distress to the sufferer, the diagnosis is changed to post-traumatic stress disorder. Post-traumatic stress disorder is further defined in DSM-IV as having three subforms: acute<sup>1</sup> (< 3 months' duration), chronic ( $\geq$  3 months' duration), and delayed onset (symptoms began at least 6 months after exposure to the trauma).

By virtue of the more sustained nature of post-

traumatic stress disorder (relative to acute stress disorder), a number of changes, including decreased self-esteem, loss of sustained beliefs about people or society, hopelessness, a sense of being permanently damaged, and difficulties in previously established relationships, are typically observed. Substance abuse often develops, especially involving alcohol, marijuana, and sedative-hypnotic drugs.

About 50 percent of cases of post-traumatic stress disorder remit within 6 months. For the remainder, the disorder typically persists for years and can dominate the sufferer's life. A longitudinal study of Vietnam veterans, for example, found 15 percent of veterans to be suffering from post-traumatic stress disorder 19 years after combat exposure (cited in McFarlane & Yehuda, 1996). In the general population, the 1-year prevalence is about 3.6 percent, with women having almost twice the prevalence of men (Kessler et al., 1995) (Table 4-1). The highest rates of post-traumatic stress disorder are found among women who are victims of crime, especially rape, as well as among torture and concentration camp survivors (Yehuda, 1999). Overall, among those exposed to extreme trauma, about 9 percent develop post-traumatic stress disorder (Breslau et al., 1998).

### **Etiology of Anxiety Disorders**

The etiology of most anxiety disorders, although not fully understood, has come into sharper focus in the last decade. In broad terms, the likelihood of developing anxiety involves a combination of life experiences, psychological traits, and/or genetic factors. The anxiety disorders are so heterogeneous that the relative roles of these factors are likely to differ. Some anxiety disorders, like panic disorder, appear to have a stronger genetic basis than others (National Institute of Mental Health [NIMH], 1998), although actual genes have not been identified. Other anxiety disorders are more rooted in stressful life events.

It is not clear why females have higher rates than males of most anxiety disorders, although some theories have suggested a role for the gonadal steroids. Other research on women's responses to stress also suggests that women experience a wider range of life

<sup>1</sup> The acute subform of post-traumatic stress disorder is distinct from acute stress disorder because the latter resolves by the end of the first month, whereas the former persists until 3 months. If the condition persists after 3 months duration, the diagnosis is again changed to the chronic post-traumatic stress disorder subform (DSM-IV).

## Mental Health: A Report of the Surgeon General

events (e.g., those happening to friends) as stressful as compared with men who react to a more limited range of stressful events, specifically those affecting themselves or close family members (Maciejewski et al., 1999).

What the myriad of anxiety disorders have in common is a state of increased arousal or fear (Barbee, 1998). Anxiety disorders often are conceptualized as an abnormal or exaggerated version of arousal. Much is known about arousal because of decades of study in animals<sup>2</sup> and humans of the so-called “fight-or-flight response,” which also is referred to as the acute stress response. The acute stress response is critical to understanding the normal response to stressors and has galvanized research, but its limitations for understanding anxiety have come to the forefront in recent years, as this section later explains.

In common parlance, the term “stress” refers either to the external stressor, which can be physical or psychosocial in nature, as well as to the internal response to the stressor. Yet researchers distinguish the two, calling the stressor the stimulus and the body’s reaction the stress response. This is an important distinction because in many anxiety states there is no immediate external stressor. The following paragraphs describe the biology of the acute stress response, as well as its limitations, in understanding human anxiety. Emerging views about the neurobiology of anxiety attempt to integrate and understand psychosocial views of anxiety and behavior in relation to the structure and function of the central and peripheral nervous system.

### **Acute Stress Response**

When a fearful or threatening event is perceived, humans react innately to survive: they either are ready for battle or run away (hence the term “fight-or-flight response”). The nature of the acute stress response is all too familiar. Its hallmarks are an almost instantaneous surge in heart rate, blood pressure, sweating, breathing, and metabolism, and a tensing of

muscles. Enhanced cardiac output and accelerated metabolism are essential for mobilizing fast action. The host of physiological changes activated by a stressful event are unleashed in part by activation of a nucleus in the brain stem called the locus ceruleus. This nucleus is the origin of most norepinephrine pathways in the brain. Neurons using norepinephrine as their neurotransmitter project bilaterally from the locus ceruleus along distinct pathways to the cerebral cortex, limbic system, and the spinal cord, among other projections.

Normally, when someone is in a serene, unstimulated state, the “firing” of neurons in the locus ceruleus is minimal. A novel stimulus, once perceived, is relayed from the sensory cortex of the brain through the thalamus to the brain stem. That route of signaling increases the rate of noradrenergic activity in the locus ceruleus, and the person becomes alert and attentive to the environment. If the stimulus is perceived as a threat, a more intense and prolonged discharge of the locus ceruleus activates the sympathetic division of the autonomic nervous system (Thase & Howland, 1995). The activation of the sympathetic nervous system leads to the release of norepinephrine from nerve endings acting on the heart, blood vessels, respiratory centers, and other sites. The ensuing physiological changes constitute a major part of the acute stress response. The other major player in the acute stress response is the hypothalamic-pituitary-adrenal axis, which is discussed in the next section.

In the 1980s, the prevailing view was that excess discharge of the locus ceruleus with the acute stress response was a major contributor to the etiology of anxiety (Coplan & Lydiard, 1998). Yet over the past decade, the limitations of the acute stress response as a model for understanding anxiety have become more apparent. The first and most obvious limitation is that the acute stress response relates to *arousal* rather than *anxiety*. Anxiety differs from arousal in several ways (Barlow, 1988; Nutt et al., 1998). First, with anxiety, the concern about the stressor is out of proportion to the realistic threat. Second, anxiety is often associated with elaborate mental and behavioral activities designed to avoid the unpleasant symptoms of a full-

---

<sup>2</sup> Anxiety is one of the few mental disorders for which animal models have been developed. Researchers can reproduce some of the symptoms of human anxiety in animals by introducing different types of stressors, either physical or psychosocial.

blown anxiety or panic attack. Third, anxiety is usually longer lived than arousal. Fourth, anxiety can occur without exposure to an external stressor.

Other limitations of this model became evident from a lack of support from clinical and basic research (Coplan & Lydiard, 1998). Furthermore, with its emphasis on the neurotransmitter norepinephrine, the model could not explain why medications that acted on the neurotransmitter serotonin (the selective serotonin reuptake inhibitors, or SSRIs) helped to alleviate anxiety symptoms. In fact, these medications are becoming the first-line treatment for anxiety disorders (Kent et al., 1998). To probe the etiology of anxiety, researchers began to devote their energies to the study of other brain circuits and the neurotransmitters on which they rely. The locus ceruleus still participates in anxiety but is understood to play a lesser role.

### ***New Views About the Anatomical and Biochemical Basis of Anxiety***

An exciting new line of research proposes that anxiety engages a wide range of neurocircuits. This line of research catapults to prominence two key regulatory centers found in the cerebral hemispheres of the brain—the hippocampus and the amygdala. These centers, in turn, are thought to activate the hypothalamic-pituitary-adrenocortical (HPA) axis<sup>3</sup> (Goddard & Charney, 1997; Coplan & Lydiard, 1998; Sullivan et al., 1998). Researchers have long established the contribution of the HPA axis to anxiety but have been perplexed by how it is regulated. They are buoyed by new findings about the roles of the hippocampus and the amygdala.

The hippocampus and the amygdala govern memory storage and emotions, respectively, among their other functions. The hippocampus is considered important in verbal memory, especially of time and place for events with strong emotional overtones (McEwen, 1998). The hippocampus and amygdala are major nuclei of the limbic system, a pathway known to

underlie emotions. There are anatomical projections between the hippocampus, amygdala, and hypothalamus (Jacobson & Sapolsky, 1991; Charney & Deutch, 1996; Coplan & Lydiard, 1998).

Studies of emotional processing in rodents (LeDoux, 1996; Rogan & LeDoux, 1996; Davis, 1997) and in humans with brain lesions (Adolphs et al., 1998) have identified the amygdala as critical to fear responses. Sensory information enters the lateral amygdala, from which processed information is passed to the central nucleus, the major output nucleus of the amygdala. The central nucleus projects, in turn, to multiple brain systems involved in the physiologic and behavioral responses to fear. Projections to different regions of the hypothalamus activate the sympathetic nervous system and induce the release of stress hormones, such as CRH.<sup>4</sup> The production of CRH in the paraventricular nucleus of the hypothalamus activates a cascade leading to release of glucocorticoids from the adrenal cortex. Projections from the central nucleus innervate different parts of the periaqueductal gray matter, which initiates descending analgesic responses (involving the body's endogenous opioids) that can suppress pain in an emergency, and which also activates species-typical defensive responses (e.g., many animals freeze when fearful).

Anxiety differs from fear in that the fear-producing stimulus is either not present or not immediately threatening, but in anticipation of danger, the same arousal, vigilance, physiologic preparedness, and negative affects and cognitions occur. Different types of internal or external factors or triggers act to produce the anxiety symptoms of panic disorder, agoraphobia, post-traumatic stress disorder, specific phobias, and generalized anxiety disorder, and the prominent anxiety that commonly occurs in major depression. It is currently a matter of research to determine whether dysregulation of these fear pathways leads to the symptoms of anxiety disorders. It has now been established, using noninvasive neuroimaging, that the human amygdala is also involved in fear responses. Fearful facial expressions have been shown to activate

<sup>3</sup> Hypothalamus and the pituitary gland, and then the cortex, or outer layer, of the adrenal gland. Upon stimulation by the pituitary hormone ACTH, the adrenal cortex releases glucocorticoids into the circulation.

<sup>4</sup> Also known as corticotropin-releasing factor.

the amygdala in MRI studies of normal human subjects (Breiter et al., 1996). Functional imaging studies in anxiety disorders, such as PET studies of brain activation in phobias (Rauch et al., 1995), are also beginning to investigate the precise neural circuits involved in the anxiety disorders.

What is especially exciting is that neuroimaging has furnished direct evidence in humans of the damaging effects of glucocorticoids. In people with post-traumatic stress disorder, neuroimaging studies have found a reduction in the size of the hippocampus. The reduced volume appears to reflect the atrophy of dendrites—the receptive portion of nerve cells—in a select region of the hippocampus. Similarly, animals exposed to chronic psychosocial stress display atrophy in the same hippocampal region (McEwen & Magarinos, 1997). Stress-induced increases in glucocorticoids are thought to be responsible for the atrophy (McEwen, 1998). If the hippocampus is impaired, the individual is thought to be less able to draw on memory to evaluate the nature of the stressor (McEwen, 1998).

### **Neurotransmitter Alterations**

There are many neurotransmitter alterations in anxiety disorders. In keeping with the broader view of anxiety, at least five neurotransmitters are perturbed in anxiety: serotonin, norepinephrine, gamma-aminobutyric acid (GABA), corticotropin-releasing hormone (CRH),<sup>5</sup> and cholecystokinin (Coplan & Lydiard 1998; Rush et al., 1998). There is such careful orchestration between these neurotransmitters that changes in one neurotransmitter system invariably elicit changes in another, including extensive feedback mechanisms. Serotonin and GABA are inhibitory neurotransmitters that quiet the stress response (Rush et al., 1998). All of these neurotransmitters have become important targets for therapeutic agents either already marketed or in development (as discussed in the section on treatment of anxiety disorders).

---

<sup>5</sup> CRH may act as a neuromodulator, a neurotransmitter, or a neurohormone, depending on the pathway.

### **Psychological Views of Anxiety**

There are several major psychological theories of anxiety: psychoanalytic and psychodynamic theory, behavioral theories, and cognitive theories (Thorn et al., 1999). Psychodynamic theories have focused on symptoms as an expression of underlying conflicts (Rush et al., 1998; Thorn et al., 1999). Although there are no empirical studies to support these psychodynamic theories, they are amenable to scientific study (Kandel, 1999) and some therapists find them useful. For example, ritualistic compulsive behavior can be viewed as a result of a specific defense mechanism that serves to channel psychic energy away from conflicted or forbidden impulses. Phobic behaviors similarly have been viewed as a result of the defense mechanism of displacement. From the psychodynamic perspective, anxiety usually reflects more basic, unresolved conflicts in intimate relationships or expression of anger.

More recent behavioral theories have emphasized the importance of two types of learning: classical conditioning and vicarious or observational learning. These theories have some empirical evidence to support them. In classical conditioning, a neutral stimulus acquires the ability to elicit a fear response after repeated pairings with a frightening (unconditioned) stimulus. In vicarious learning, fearful behavior is acquired by observing others' reactions to fear-inducing stimuli (Thorn et al., 1999). With general anxiety disorder, unpredictable positive and negative reinforcement is seen as leading to anxiety, especially because the person is unsure about whether avoidance behaviors are effective.

Cognitive factors, especially the way people interpret or think about stressful events, play a critical role in the etiology of anxiety (Barlow et al., 1996; Thorn et al., 1999). A decisive factor is the individual's perception, which can intensify or dampen the response. One of the most salient negative cognitions in anxiety is the sense of uncontrollability. It is typified by a state of helplessness due to a perceived inability to predict, control, or obtain desired results (Barlow et al., 1996). Negative cognitions are frequently found in individuals with anxiety (Ingram et al., 1998). Many

modern psychological models of anxiety incorporate the role of individual vulnerability, which includes both genetic (Smoller & Tsuang, 1998) and acquired (Coplan et al., 1997) predispositions. There is evidence that women may ruminate more about distressing life events compared with men, suggesting that a cognitive risk factor may predispose them to higher rates of anxiety and depression (Nolen-Hoeksema et al., in press).

### **Treatment of Anxiety Disorders**

The anxiety disorders are treated with some form of counseling or psychotherapy or pharmacotherapy, either singly or in combination (Barlow & Lehman, 1996; March et al., 1997; American Psychiatric Association, 1998; Kent et al., 1998).

### ***Counseling and Psychotherapy***

Anxiety disorders are responsive to counseling and to a wide variety of psychotherapies. More severe and persistent symptoms also may require pharmacotherapy (American Psychiatric Association, 1998).

During the past several decades, there has been increasing enthusiasm for more focused, time-limited therapies that address ways of coping with anxiety symptoms more directly rather than exploring unconscious conflicts or other personal vulnerabilities (Barlow & Lehman, 1996). These therapies typically emphasize cognitive and behavioral assessment and interventions.

The hallmarks of cognitive-behavioral therapies are evaluating apparent cause and effect relationships between thoughts, feelings, and behaviors, as well as implementing relatively straightforward strategies to lessen symptoms and reduce avoidant behavior (Barlow, 1988). A critical element of therapy is to increase exposure to the stimuli or situations that provoke anxiety. Without such therapeutic assistance, the sufferer typically withdraws from anxiety-inducing situations, inadvertently reinforcing avoidant or escape behavior.

The therapist provides reassurance that the feared situation is not deadly and introduces a plan to enhance mastery. This plan may include approaching the feared situation in a graduated or stepwise hierarchy or teaching the patient to use responses that dampen anxiety, such as deep muscle relaxation or coping. One fundamental principle is that prolonged exposure to a feared stimulus reliably decreases cognitive and physiologic symptoms of anxiety (Marks, 1969; Barlow, 1988). With such experience generally comes greater self-efficacy and a greater willingness to encounter other feared stimuli. For panic disorder, interoceptive training (a type of conditioning technique) and breathing exercises are often employed to help the sufferer become more capable of recognizing and coping with the social cues, antecedents, or early signs of a panic attack. Cognitive interventions are used to counteract the exaggerated or catastrophic thoughts that characterize anxiety. For treatment of obsessive-compulsive disorder, the strategy of response prevention must be added to exposure to ensure that compulsions are not performed (Barlow, 1988).

There is now extensive evidence that cognitive-behavioral therapies are useful treatments for a majority of patients with anxiety disorders (Chambless et al., 1998). Poorer outcomes are observed, however, in more complicated patient groups. With obsessive-compulsive disorder, approximately 20 to 25 percent of patients are unwilling to participate in therapy (March et al., 1997). Another major limitation of cognitive-behavioral therapies is not their effectiveness but, rather, the limited availability of skilled practitioners (Ballenger et al., 1998).

It is possible that more traditional forms of therapy based on psychodynamic or interpersonal theories of anxiety also may prove to be effective treatments (Shear, 1995). However, these therapies have not yet received extensive empirical support. As a result, more traditional therapies are generally deemphasized in evidence-based treatment guidelines for anxiety disorders.



## Mental Health: A Report of the Surgeon General

### *Pharmacotherapy*

The medications typically used to treat patients with anxiety disorders are benzodiazepines, antidepressants, and the novel compound buspirone (Lydiard et al., 1996). In light of increasing awareness of numerous neurochemical alterations in anxiety disorders, many new classes of drugs are likely to be developed, expressly targeting CRH and other neuroactive agents (Nemeroff, 1998).

#### *Benzodiazepines*

The benzodiazepines are a large class of relatively safe and widely prescribed medications that have rapid and profound antianxiety and sedative-hypnotic effects. The benzodiazepines are thought to exert their therapeutic effects by enhancing the inhibitory neurotransmitter systems utilizing GABA. Benzodiazepines bind to a site on the GABA receptor and act as receptor agonists (Perry et al., 1997). Benzodiazepines differ in terms of potency, pharmacokinetics (i.e., elimination half-life), and lipid solubility.

The four benzodiazepines currently widely prescribed for treatment of anxiety disorders are diazepam, lorazepam, clonazepam, and alprazolam. Each is now available in generic formulations (Davidson, 1998). Among these agents, alprazolam and lorazepam have shorter elimination half-lives—that is, are removed from the body more quickly—while diazepam and clonazepam have a long period of action (i.e., up to 24 hours). Diazepam also has multiple active metabolites, which increase the risk of “carryover” effects such as sedation and “hangover.” Benzodiazepines that undergo conjugation appear to have longer elimination time in women, and oral contraceptive can decrease clearance (Dawkins, 1995). Since Asians are more likely to metabolize diazepam more slowly, they may require lower doses to achieve the same blood concentrations as Caucasians (Lin et al., 1997).

Benzodiazepines have the potential for producing drug dependence (i.e., physiological or behavioral symptoms after discontinuation of use). Shorter acting compounds have somewhat greater liability because of more rapid and abrupt onset of withdrawal symptoms.

Because the benzodiazepines do not have strong antiobsessional effects, their use in obsessive-compulsive disorder and post-traumatic stress disorder is generally viewed as palliative (i.e., relieving, but not eliminating symptoms). Rather, obsessive-compulsive disorder and post-traumatic stress disorder are more effectively treated by antidepressants, especially the SSRIs (as discussed below). When effective, benzodiazepines should be tapered after several months of use, although there is a substantial risk of relapse. Many clinicians favor a combined treatment approach for panic disorder and generalized anxiety disorder, in which benzodiazepines are used acutely in tandem with an antidepressant. The benzodiazepines are subsequently tapered as the antidepressant’s therapeutic effects begin to emerge (American Psychiatric Association, 1998).

#### *Antidepressants*

Most antidepressant medications have substantial antianxiety and antipanic effects in addition to their antidepressant action (Kent et al., 1998). Moreover, a large number of antidepressants have antiobsessional effects (Perry et al., 1997). The observation that the tricyclic antidepressant imipramine had a different anxiolytic profile than diazepam helped to differentiate panic disorder from generalized anxiety disorder and, subsequently, social phobia.

Clomipramine, a tricyclic antidepressant (TCA) with relatively potent reuptake inhibitory effects on serotonin (5-HT) neurons, subsequently was found to be the only TCA to have specific antiobsessional effects (March et al., 1997). The importance of this effect on 5-HT was highlighted when the SSRIs became available. By the late 1990s, it became clear that all of the SSRIs have antiobsessional effects (Greist et al., 1995; Kent et al., 1998).

Current practice guidelines rank the TCAs below the SSRIs for treatment of anxiety disorders because of the SSRIs’ more favorable tolerability and safety profiles (March et al., 1997; American Psychiatric Association, 1998; Ballenger et al., 1998;). Nevertheless, there are patients who respond to the TCAs after failing to respond to one or more of the

newer agents. Similarly, although relatively rarely used, the monoamine oxidate inhibitors (MAOIs) have significant antiobsessional, antipanic, and anxiolytic effects (Sheehan et al., 1980; American Psychiatric Association, 1998). In the United States, the MAOIs phenelzine, tranylcypromine, and isocarboxazid (which has not been consistently marketed this decade) are seldom used unless simpler medication strategies have failed (American Psychiatric Association, 1998).

The five drugs within the SSRI class—fluoxetine, sertraline, paroxetine, fluvoxamine, and citalopram—have emerged as the preferred type of antidepressant for treatment of anxiety disorders (Westenberg, 1996; Kent et al., 1998). In addition to well-established efficacy in obsessive-compulsive disorder, there is convincing and growing evidence of antipanic and broader anxiolytic effects (American Psychiatric Association, 1998; Kent et al., 1998). Treatment of panic disorder often requires lower initial doses and slower upward titration. By contrast, treatment for obsessive-compulsive disorder ultimately may entail higher doses (for example, 60 or 80 mg/day of fluoxetine or 200 mg per day of sertraline) and longer durations to achieve desired outcomes (March et al., 1997). As all of the SSRIs are currently protected by patents, there are no generic forms yet available. This adds to the direct costs of treatment. Cost may be offset indirectly, however, by virtue of need for fewer treatment visits and fewer concomitant medications, and cost likely will abate when these agents begin to lose patent protection in a few years.

Other newer antidepressants, including venlafaxine, nefazodone, and mirtazapine, also may have significant antianxiety effects, for which clinical trials are under way (March et al., 1997; American Psychiatric Association, 1998). Paroxetine has been approved by the Food and Drug Administration (FDA) for social phobia, and sertraline is being developed for post-traumatic stress disorder. Nefazodone, which also is being studied in post-traumatic stress disorder, and mirtazapine may possess lower levels of sexual side effects, a problem that complicates longer term treatment with SSRIs, venlafaxine, TCAs, and MAOIs (Baldwin & Birtwistle, 1998).

When effective in treating anxiety, antidepressants should be maintained for at least 4 to 6 months, then tapered slowly to avoid discontinuation-emergent activation of anxiety symptoms (March et al., 1997; American Psychiatric Association, 1998; Ballenger et al., 1998). Although less extensively researched than depression, it is likely that many patients with anxiety disorders may warrant longer term, indefinite treatment to prevent relapse or chronicity.

### *Buspirone*

This azopyrine compound is a relatively selective 5-HT<sub>1A</sub> partial agonist (Stahl, 1996). It was approved by the FDA in the mid-1980s as an anxiolytic. However, unlike the benzodiazepines, buspirone is not habit forming and has no abuse potential. Buspirone also has a safety profile comparable to the SSRIs, and it is significantly better tolerated than the TCAs.

Buspirone does not block panic attacks, and it is not efficacious as a primary treatment of obsessive-compulsive disorder or post-traumatic stress disorder (Stahl, 1996). Buspirone is most useful for treatment of generalized anxiety disorder, and it is now frequently used as an adjunct to SSRIs (Lydiard et al., 1996). Buspirone takes 4 to 6 weeks to exert therapeutic effects, like antidepressants, and it has little value for patients when taken on an "as needed" basis.

### ***Combinations of Psychotherapy and Pharmacotherapy***

Some patients with anxiety disorders may benefit from both psychotherapy and pharmacotherapy treatment modalities, either combined or used in sequence (March et al., 1997; American Psychiatric Association, 1998). Drawing from the experiences of depression researchers, it seems likely that such combinations are not uniformly necessary and are probably more cost-effective when reserved for patients with more complex, complicated, severe, or comorbid disorders. The benefits of multimodal therapies for anxiety need further study.

## Mental Health: A Report of the Surgeon General

### Mood Disorders

In 1 year, about 7 percent of Americans suffer from mood disorders, a cluster of mental disorders best recognized by depression or mania (Table 4-1). Mood disorders are outside the bounds of normal fluctuations from sadness to elation. They have potentially severe consequences for morbidity and mortality.

This section covers four mood disorders. As the predominant mood disorder, major depressive disorder (also known as unipolar major depression), garners the greatest attention. It is twice more common in women than in men, a gender difference that is discussed later in this section. The other mood disorders covered below are bipolar disorder, dysthymia, and cyclothymia.

Mood disorders rank among the top 10 causes of worldwide disability (Murray & Lopez, 1996). Unipolar major depression ranks first, and bipolar disorder ranks in the top 10. Moreover, disability and suffering are not limited to the patient. Spouses, children, parents, siblings, and friends experience frustration, guilt, anger, financial hardship, and, on occasion, physical abuse in their attempts to assuage or cope with the depressed person's suffering. Women between the ages of 18 and 45 comprise the majority of those with major depression (Regier et al., 1993).

Depression also has a deleterious impact on the economy, both in diminished productivity and in use of health care resources (Greenberg et al., 1993). In the workplace, depression is a leading cause of absenteeism and diminished productivity. Although only a minority seek professional help to relieve a mood disorder, depressed people are significantly more likely than others to visit a physician for some other reason. Depression-related visits to physicians thus account for a large portion of health care expenditures. Seeking another or a less stigmatized explanation for their difficulties, some depressed patients undergo extensive and expensive diagnostic procedures and then get treated for various other complaints while the mood disorder goes undiagnosed and untreated (Wells et al., 1989).

### Complications and Comorbidities

Suicide is the most dreaded complication of major depressive disorders. About 10 to 15 percent of patients formerly hospitalized with depression commit suicide (Angst et al., 1999). Major depressive disorders account for about 20 to 35 percent of all deaths by suicide (Angst et al., 1999). Completed suicide is more common among those with more severe and/or psychotic symptoms, with late onset, with co-existing mental and addictive disorders (Angst et al., 1999), as well as among those who have experienced stressful life events, who have medical illnesses, and who have a family history of suicidal behavior (Blumenthal, 1988). In the United States, men complete suicide four times as often as women; women attempt suicide four times as frequently as do men (Blumenthal, 1988). Recognizing the magnitude of this public health problem, the Surgeon General issued a *Call to Action on Suicide* in 1999 (see Figure 4-1). Individuals with depression also face an increased risk of death from coronary artery disease (Glassman & Shapiro, 1998).

Mood disorders often coexist, or are *comorbid*, with other mental and somatic disorders. Anxiety is commonly comorbid with major depression. About one-half of those with a primary diagnosis of major depression also have an anxiety disorder (Barbee, 1998; Regier et al., 1998). The comorbidity of anxiety and depression is so pronounced that it has led to theories of similar etiologies, which are discussed below. Substance use disorders are found in 24 to 40 percent of individuals with mood disorders in the United States (Merikangas et al., 1998). Without treatment, substance abuse worsens the course of mood disorders. Other common comorbidities include personality disorders (DSM-IV) and medical illness, especially chronic conditions such as hypertension and arthritis. People with depression have a high prevalence (65 to 71 percent) of any of eight common chronic medical conditions (Wells et al., 1991). The mood disorders also may alter or "scar" personality development.

**Figure 4-1. Surgeon General's Call to Action to Prevent Suicide—1999**

- Suicide is a serious public health problem
  - 31,000 suicides in 1996
  - 500,000 people visit emergency rooms due to attempted suicide
- Suicide rate declined from 12.1 per 100,000 in 1976 to 10.8 per 100,000 in 1996
  - Rate in adolescents and young adults almost tripled since 1952
  - Rate is 50 percent higher than the homicide rate
- National Strategy for Suicide Prevention: AIM
  - Awareness: promote public awareness of suicide as a public health problem
  - Intervention: enhance services and programs
  - Methodology: advance the science of suicide prevention
- Risk factors
  - Male gender
  - Mental disorders, particularly depression and substance abuse
  - Prior suicide attempts
  - Unwillingness to seek help because of stigma
  - Barriers to accessing mental health treatment
  - Stressful life event/loss
  - Easy access to lethal methods such as guns
- Protective factors
  - Effective and appropriate clinical care for underlying disorders
  - Easy access to care
  - Support from family, community, and health and mental health care staff

### ***Clinical Depression Versus Normal Sadness***

People have been plagued by disorders of mood for at least as long as they have been able to record their experiences. One of the earliest terms for depression, “melancholy,” literally meaning “black bile,” dates back to Hippocrates. Since antiquity, dysphoric states outside the range of normal sadness or grief have been recognized, but only within the past 40 years or so have researchers had the means to study the changes in cognition and brain functioning that are associated with severe depressive states.

At some time or another, virtually all adult human beings will experience a tragic or unexpected loss, romantic heartbreak, or a serious setback and times of profound sadness, grief, or distress. Indeed, something is awry if the usual expressions of sadness do not accompany such situations so common to the human condition—death of a loved one, severe illness, prolonged disability, loss of employment or social status, or a child’s difficulties, for example.

What is now called major depressive disorder, however, differs both *quantitatively* and *qualitatively* from normal sadness or grief. Normal states of dysphoria (a negative or aversive mood state) are typically less pervasive and generally run a more time-limited course. Moreover, some of the symptoms of severe depression, such as anhedonia (the inability to experience pleasure), hopelessness, and loss of mood reactivity (the ability to feel a mood uplift in response to something positive) only rarely accompany “normal” sadness. Suicidal thoughts and psychotic symptoms such as delusions or hallucinations virtually always signify a pathological state.

Nevertheless, many other symptoms commonly associated with depression are experienced during times of stress or bereavement. Among them are sleep disturbances, changes in appetite, poor concentration, and ruminations on sad thoughts and feelings. When a person suffering such distress seeks help, the diagnostician’s task is to differentiate the normal from the pathologic and, when appropriate, to recommend treatment.

### **Assessment: Diagnosis and Syndrome Severity**

The criteria for diagnosing major depressive episode, dysthymia, mania, and cyclothymia are presented in Tables 4-2 through 4-5. Mania is an essential feature of bipolar disorder, which is marked by episodes of mania or mixed episodes of mania and depression. The reliability of the diagnostic criteria for major depressive disorder and bipolar disorder is impressive, with greater than 90 percent agreement reached by independent evaluators (DSM-IV).

## Mental Health: A Report of the Surgeon General

### **Major Depressive Disorder**

Major depressive disorder features one or more major depressive episodes (see Table 4-2), each of which lasts at least 2 weeks (DSM-IV). Since these episodes are also characteristic of bipolar disorder, the term “major<sup>6</sup> depression” refers to both major depressive disorder and the depression of bipolar disorder.

The cardinal symptoms of major depressive disorder are depressed mood and loss of interest or pleasure. Other symptoms vary enormously. For example, insomnia and weight loss are considered to be classic signs, even though many depressed patients gain weight and sleep excessively. Such heterogeneity is partly dealt with by the use of diagnostic subtypes (or course modifiers) with differing presentations and prevalence. For example, a more severe depressive syndrome characterized by a constellation of classical signs and symptoms, called melancholia, is more common among older than among younger people, as are depressions characterized by psychotic features (i.e., delusions and hallucinations) (DSM-IV). In fact, the presentation of psychotic features without concomitant melancholia should always raise suspicion about the accuracy of the diagnosis (*vis-à-vis* schizophrenia or a related psychotic disorder). The so-called *reversed vegetative symptoms* (oversleeping, overeating, and weight gain) may be more prevalent in women than men (Nemeroff, 1992). Anxiety symptoms such as panic attacks, phobias, and obsessions also are not uncommon.

When untreated, a major depressive episode may last, on average, about 9 months. Eighty to 90 percent of individuals will remit within 2 years of the first episode (Kapur & Mann, 1992). Thereafter, at least 50 percent of depressions will recur, and after three or more episodes the odds of recurrence within 3 years increases to 70 to 80 percent if the patient has not had preventive treatment (Thase & Sullivan, 1995). Thus, for many, an initial episode of major depression will evolve over time into the more recurrent illness

sometimes referred to as unipolar major depression (Thase & Sullivan, 1995). Each new episode also confers new risks of chronicity, disability, and suicide.

### **Dysthymia**

Dysthymia is a chronic form of depression. Its early onset and unrelenting, “smoldering” course are among the features that distinguish it from major depressive disorder (DSM-IV). Dysthymia becomes so intertwined with a person’s self-concept or personality that the individual may be misidentified as “neurotic” (resulting from unresolved early conflicts expressed through unconscious personality defenses or characterologic disorders) (Akiskal, 1985). Indeed, the onset of dysthymia in childhood or adolescence undoubtedly affects personality development and coping styles, particularly prompting passive, avoidant, and dependent “traits.” To avoid the pejorative connotations associated with the terms “neurotic” and “characterologic,” the term “dysthymia” is used in DSM-IV as a descriptive, or atheoretical, diagnosis for a chronic form of depression (see Table 4-3) (DSM-IV). Affecting about 2 percent of the adult population in 1 year, dysthymia is defined by its subsyndromal nature (i.e., fewer than the five persistent symptoms required to diagnose a major depressive episode) and a protracted duration of at least 2 years for adults and 1 year for children. Like other early-onset disorders, dysthymic disorder is associated with higher rates of comorbid substance abuse. People with dysthymia also are susceptible to major depression. When this occurs, their illness is sometimes referred to as “double depression,” that is, the combination of dysthymia and major depression (Keller & Shapiro, 1982). Unlike the superimposed major depressive episode, however, the underlying dysthymia seldom remits spontaneously. Women are twice as likely to be diagnosed with dysthymia as men (Robins & Regier, 1991).

### **Bipolar Disorder**

Bipolar disorder is a recurrent mood disorder featuring one or more episodes of mania or mixed episodes of mania and depression (DSM-IV; Goodwin & Jamison,

---

<sup>6</sup> The adjective “major” before the word “depression” denotes the number of symptoms required for the diagnosis, as distinct, from a proposed new category of “minor depression,” which requires fewer symptoms (see Chapter 5).

Table 4-2. DSM-IV criteria for major depressive episode

- A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.
- Note:** Do not include symptoms that are clearly due to a general medical condition, or mood-incongruent delusions or hallucinations.
- (1) depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad or empty) or observation made by others (e.g., appears tearful). **Note:** In children and adolescents, can be irritable mood.
  - (2) markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation made by others).
  - (3) significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. **Note:** In children, consider failure to make expected weight gains.
  - (4) insomnia or hypersomnia nearly every day.
  - (5) psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
  - (6) fatigue or loss of energy nearly every day.
  - (7) feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
  - (8) diminished ability to think or concentrate, or indecisiveness, nearly every day (either subjective account or as observed by others).
  - (9) recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.
- B. The symptoms do not meet criteria for a mixed episode.
- C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- D. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hypothyroidism).
- E. The symptoms are not better accounted for by bereavement, i.e., after the loss of a loved one; the symptoms persist for longer than 2 months or are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.

## Mental Health: A Report of the Surgeon General

**Table 4-3. DSM-IV diagnostic criteria for Dysthymic Disorder**

- A. Depressed mood for most of the day, for more days than not, as indicated either by subjective account or observation by others, for at least 2 years. **Note:** In children and adolescents, mood can be irritable and duration must be at least 1 year.
- B. Presence, while depressed, of two (or more) of the following:
  - (1) poor appetite or overeating
  - (2) insomnia or hypersomnia
  - (3) low energy or fatigue
  - (4) low self-esteem
  - (5) poor concentration or difficulty making decisions
  - (6) feelings of hopelessness
- C. During the 2-year period (1 year for children or adolescents) of the disturbance, the person has never been without the symptoms in Criteria A and B for more than 2 months at a time.
- D. No major depressive episode has been present during the first 2 years of the disturbance (1 year for children and adolescents); i.e., the disturbance is not better accounted for by chronic major depressive disorder, or major depressive disorder, in partial remission.

**Note:** There may have been a previous major depressive episode provided there was a full remission (no significant signs or symptoms for 2 months) before development of the dysthymic disorder. In addition, after the initial 2 years (1 year in children or adolescents) of dysthymic disorder, there may be superimposed episodes of major depressive disorder, in which case both diagnoses may be given when the criteria are met for a major depressive episode.
- E. There has never been a manic episode, a mixed episode, or a hypomanic episode, and criteria have never been met for cyclothymic disorder.
- F. The disturbance does not occur exclusively during the course of a chronic psychotic disorder, such as schizophrenia or delusional disorder.
- G. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hypothyroidism).
- H. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

1990). Bipolar disorder<sup>7</sup> is distinct from major depressive disorder by virtue of a history of manic or hypomanic (milder and not psychotic) episodes. Other differences concern the nature of depression in bipolar disorder. Its depressive episodes are typically associated with an earlier age at onset, a greater likelihood of reversed vegetative symptoms, more frequent episodes or recurrences, and a higher familial prevalence (DSM-IV; Goodwin & Jamison, 1990). Another noteworthy difference between bipolar and nonbipolar groups is the differential therapeutic effect of lithium salts, which are more helpful for bipolar disorder (Goodwin & Jamison, 1990).

Mania is derived from a French word that literally means crazed or frenzied. The mood disturbance can range from pure euphoria or elation to irritability to a labile admixture that also includes dysphoria (Table 4-4). Thought content is usually grandiose but also can be paranoid. Grandiosity usually takes the form both of overvalued ideas (e.g., "My book is the best one ever written") and of frank delusions (e.g., "I have radio transmitters implanted in my head and the Martians are monitoring my thoughts.") Auditory and visual hallucinations complicate more severe episodes. Speed of thought increases, and ideas typically race through the manic person's consciousness. Nevertheless, distractibility and poor concentration commonly impair implementation. Judgment also can be severely compromised; spending sprees, offensive or disinhibited behavior, and promiscuity or other objectively reckless behaviors are commonplace. Subjective energy, libido, and activity typically increase but a perceived reduced need for sleep can sap physical reserves. Sleep deprivation also can exacerbate cognitive difficulties and contribute to development of catatonia or a florid, confusional state known as delirious mania. If the manic patient is delirious, paranoid, or catatonic, the behavior is difficult to distinguish from that of a schizophrenic patient. Clinicians are prone to misdiagnose mania as schizophrenia in African Americans (Bell & Mehta,

1981). Most people with bipolar disorder have a history of remission and at least satisfactory functioning before onset of the index episode of illness.

In DSM-IV, bipolar depressions are divided into type I (prior mania) and type II (prior hypomanic episodes only). About 1.1 percent of the adult population suffers from the type I form, and 0.6 percent from the type II form (Goodwin & Jamison, 1990; Kessler et al., 1994) (Table 4-5). Episodes of mania occur, on average, every 2 to 4 years, although accelerated mood cycles can occur annually or even more frequently. The type I form of bipolar disorder is about equally common in men and women, unlike major depressive disorder, which is more common in women.

Hypomania, as suggested above, is the sub-syndromal counterpart of mania (DSM-IV; Goodwin & Jamison, 1990). By definition, an episode of hypomania is never psychotic nor are hypomanic episodes associated with marked impairments in judgment or performance. In fact, some people with bipolar disorder long for the productive energy and heightened creativity of the hypomanic phase.

Hypomania can be a transitional state (i.e., early in an episode of mania), although at least 50 percent of those who have hypomanic episodes never become manic (Goodwin & Jamison, 1990). Whereas a majority have a history of major depressive episodes (bipolar type II disorder), others become hypomanic only during antidepressant treatment (Goodwin & Jamison, 1990). Despite the relatively mild nature of hypomania, the prognosis for patients with bipolar type II disorder is poorer than that for recurrent (unipolar) major depression, and there is some evidence that the risk of rapid cycling (four or more episodes each year) is greater than with bipolar type I (Coryell et al., 1992). Women are at higher risk for rapid cycling bipolar disorder than men (Coryell et al., 1992). Women with bipolar disorder are also at increased risk for an episode during pregnancy and the months following childbirth (Blehar et al., 1998).

<sup>7</sup> Bipolar disorder is also known as bipolar affective disorder and manic depression.



## Mental Health: A Report of the Surgeon General

**Table 4-4. DSM-IV criteria for manic episode**

- A. A distinct period of abnormally and persistently elevated, expansive, or irritable mood, lasting at least 1 week (or any duration if hospitalization is necessary).
- B. During the period of mood disturbance, three (or more) of the following symptoms have persisted (four if the mood is only irritable) and have been present to a significant degree:
  - (1) inflated self-esteem or grandiosity
  - (2) decreased need for sleep (e.g., feels rested after only 3 hours of sleep)
  - (3) more talkative than usual or pressure to keep talking
  - (4) flight of ideas or subjective experience that thoughts are racing
  - (5) distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli)
  - (6) increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation
  - (7) excessive involvement in pleasurable activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments)
- C. The symptoms do not meet criteria for a mixed episode.
- D. The mood disturbance is sufficiently severe to cause marked impairment in occupational functioning or in usual social activities or relationships with others, or to necessitate hospitalization to prevent harm to self or others, or there are psychotic features.
- E. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication, or other treatment) or general medical condition (e.g., hyperthyroidism).

**Note:** Manic-like episodes that are clearly caused by somatic antidepressant treatment (e.g., medication, electroconvulsive therapy, light therapy) should not count toward a diagnosis of bipolar I disorder.

**Table 4-5. DSM-IV diagnostic criteria for Cyclothymic Disorder**

- A. For at least 2 years, the presence of numerous periods with hypomanic symptoms and numerous periods with depressive symptoms that do not meet criteria for a major depressive episode. **Note:** In children and adolescents, the duration must be at least 1 year.
- B. During the above 2-year period (1 year in children and adolescents), the person has not been without the symptoms in Criterion A for more than 2 months at a time.
- C. No major depressive episode, manic episode, or mixed episode has been present during the first 2 years of the disturbance.  
  
**Note:** After the initial 2 years (1 year in children and adolescents) of cyclothymic disorder, there may be superimposed manic or mixed episodes (in which case both bipolar I disorder and cyclothymic disorder may be diagnosed) or major depressive episodes (in which case both bipolar II disorder and cyclothymic disorder may be diagnosed).
- D. The symptoms in Criterion A are not better accounted for by schizoaffective disorder and are not superimposed on schizophrenia, schizophreniform disorder, delusional disorder, or psychotic disorder not otherwise specified.
- E. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hyperthyroidism).
- F. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.